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Analysis of the Vertical Facial Form in Patients With Severe Hypodontia

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ABSTRACT We examined the lateral cephalograms of Russian patients in the following categories: control with acceptable occlusions (group 1); severe hypodontia with absence of six or more teeth (group 2); and severe hypodontia associated with hypohidrotic ectodermal dysplasia (HED) (group 3). Analysis was in a cross-sectional manner, comparing dimensions at the start of the mixed dentition phase (age 6–10) and in the permanent dentition (age 12–18). The groups were matched for age and sex. Thirty-one hard- and soft-tissue landmarks were traced, and 35 linear, 19 angular, and 7 ratioed measurements were taken and compared, using analysis of variance to compare the means of each group.

A reduced anterior face height was found in groups 2 and 3 as a consequence of a reduced anterior *lower* face height. In group 2 in the mixed dentition, the posterior face height was also reduced. The inclination of the mandible (<Se S Go Gn) was significantly reduced to 28.22° ± 0.71° in group 2 and to 24.07° ± 0.97° in group 3. The facial profile appeared flat or concave (<se pn pg was increased up to 8.42° ± 1.56° in children and 16.81° ± 2.18° in adolescents). The subnasion point was behind the aesthetic line (EL), and in group 2 patients the naso-labial angle was obtuse when compared to nonaffected patients. In group 3 patients, the naso-labial angle became acute and lips were protuberant and everted as a consequence of the reduced vertical height. Groups 2 and 3 have the typical facial characteristics unique to hypodontia, with reduced vertical dimensions as a consequence of limited alveolar bone growth. However, group 3 patients have a unique abnormal craniofacial development. Am J Phys Anthropol 111:177–184, 2000.

In this investigation we have defined severe hypodontia as the congenital absence of six or more permanent teeth, excluding the third molars. The multiple absence of numerous teeth can occur either in isolation or in association with other clinical signs as a syndrome (oligodontia). The general consequences on long-term dental management are often profound, requiring protracted care involving many dental specialties. It is recognized that certain patterns of hypodontia are genetically determined, and the importance of genetic specificity has been examined, with some groups identifying mutations in MSX1 as a cause of dental agenesis (Vastardis et al., 1996), while others have questioned its role (Niemenen et al., 1995). Whatever the cause, it is believed that other features of dental development are affected, suggesting a more widespread effect of the

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abnormality. For example, it is known that tooth size varies in patients with oligodontia, with a general reduction in dental dimensions reflecting the generalized consequences of this defect in development (Schalk-Van der Weide and Bosman, 1996). While these effects can be seen to be more widespread than the simple agenesis of tooth formation, it is unclear if the possible defects of the condition can affect growth of the facial skeleton. The literature is very sparse on this aspect of oligodontia (Bixler et al., 1988).

A significant clinical syndrome associated with severe hypodontia is ectodermal dysplasia or hypohydrotic ectodermal dysplasia (HED), where there is an abnormality of the development of ectodermal tissues. In these cases, patients not only demonstrate a reduction in the number of teeth, but also show a reduction in the number of sweat glands and have thin, sparse hair (Nakata et al., 1980).

In addition, it is well-known that the facial skeleton varies not only between species but also between ethnic groups, and has changed during evolution. Furthermore, the changes of mandibular shape during evolution are associated with a reduction in the number of teeth. While the overall reduction in facial skeletal dimensions is thought to be due mainly to a change in the consistency of our diet (Kaifu, 1997), the effects of gene interactions between ethnic groups must also add to this evolutionary change, as demonstrated in nonhuman primates (Ravosa, 1996). The general characteristics of the facial skeleton of extant monkeys show an increase in length of snout, an anteriorly tapering maxilla, and a large facial height below the orbits (Benefit and McCrossin, 1991).

Therefore, our aim was to examine the cross-sectional radiographic data of normative Russian patients and to compare these to patients with severe hypodontia in isolation and hypodontia in association with hypohidrotic ectodermal dysplasia. The specific null hypothesis we wished to examine was that there was no difference in the vertical facial skeleton in nonsyndromic and syndromic patients when compared to unaffected Russian counterparts. We wished to establish if the alveolar bone of the maxilla and mandible contributed significantly to growth in the vertical dimension of the craniofacial structures, and that as a consequence of a reduction in the number of teeth, there is also a reduction in facial height.

MATERIALS AND METHODS

Orthopantomogram radiographs of 1,516 patients aged 6–18 years, presenting with malocclusions, were used to establish the occurrence of the condition of hypodontia. Lateral skull radiographs were taken before active treatment of all patients, and they were divided into:

Group 1. A clinically acceptable occlusion (class I) with no apparent abnormality of facial appearance, i.e., class I or mild class II skeletal base with a class I incisor relationship; the lips were on or close to the E line (n = 63; 31 in the mixed dentition, and 32 in the permanent dentition).

Group 2. An abnormal occlusion with six or more congenitally absent missing teeth. The sweat gland function was established to be within normal limits, and there appeared to be normal hair distribution (n = 85; 40 in the mixed dentition, and 45 in the permanent dentition). Those patients with abnormal sweat gland function and a reduction in hair were allocated to group 3; they were diagnosed as having syndromic hypohidrotic ectodermal dysplasia.

Group 3. Severe hypodontia as part of hypohydrotic ectodermal dysplasia (n = 34; 15 in the mixed dentition, and 19 in the permanent dentition).

Group 4. This group was examined to establish the overall pattern of hypodontia in a Russian population. In this group, which consisted of 1,516 orthopantomograms taken as a screen of patients attending a school clinic, 256 patients (16.9%) had congenitally missing teeth.

The patients of groups 2 and 3 were referred to another specialist center for treatment.

Each group age varied in that in the mixed dentition, the age spanned 6–10 years, and in the permanent dentition the age was 12–18 years.

Thirty-one hard- and soft-tissue landmarks were identified and traced on each cephalogram. This allowed analysis of 35 linear, 19 angular, and 7 ratioed measurements (Björk, 1947; Downs, 1948; Ricketts, 1969; Biggerstaff et al., 1977). The specific analysis is outlined in Figure 1, showing the detailed labeling of points, while Table 1a,b lists their definitions.

Analysis of variance with repeated measures was used to compare the changes in a cross-sectional analysis between groups. In addition, each group was also compared independently, using multiple comparisons, with a Bonferroni correction for multiple Student's *t*-test ($P < 0.01$). This eliminated the findings by chance with multiple comparisons (Bland, 1995).

RESULTS

Table 2 outlines the breakdown of teeth which were absent, and clearly shows this to be similar to reported Caucasian patterns of absence (Maklin et al., 1979). In the major group of this study in which nonsyndromic hypodontia patients were compared to "normal" and ectodermal dysplasia patients, 182 patients were identified who had complete records, including lateral skull radiographs.

The typical lateral skull radiographs are shown of two patients who were clinically diagnosed with severe hypodontia. The first (age 21 years) has clinically diagnosed hypodontia with the absence of 28 permanent teeth, but without any systemic defects (Fig. 2a); the second case (age 23) has 29 missing teeth but has the clinical signs of syndromic hypohidrotic ectodermal dysplasia (Fig. 2b). Notice in both cases the apparent lack of alveolar bone, with a reduced lower face height.

One hundred and nineteen patients (7.85%) presented with severe hypodontia, with 34 (2.24%) of them diagnosed as suffering from HED. Sixty-three patients were used to compare with both groups as a nonaffected group. Only the measurements with significant differences are reported here; all data were compared (Tables 3–5).

The markedly decreased anterior face height (Se-Me) in hypodontia groups 2 and 3 was mainly due to the significantly shorter anterior lower facial height (ANS-Me) (Table 5). In the group 2 patients with mixed dentition, the posterior facial height (S-Go) also decreased (Table 3). The patients with HED had a reduced general facial divergence as a result of the prevalent decrease of the anterior facial height (Se-Me) over the lack in the posterior facial height (SGo). In the period of permanent dentition, the posterior facial height (S-Go) increased in the hypodontia groups (2 and 3) and did not indicate any deviation from normal development. The type of face was hypo-divergent due to a considerable decrease of the anterior facial height. The inclination of the mandible (<Se S Go Gn) was significantly reduced up to $28.22° \pm 0.71°$ in hypodontia group 2 and $24.07° \pm 0.97°$ in HED group 3. The dimensions of the mid-face in group 2 were equal to those in the control group. While the anterior upper facial height (Se-ANS: Se-SpP) was initially reduced in the mixed dentition phase, following growth the value approximated that of nonaffected cases.

Craniofacial mid-face characteristics of the patients with hypohidrotic ectodermal dysplasia (group 3) displayed a lack of upper anterior (Se-ANS) and posterior (CRF-PSeS) facial heights (Table 4). The ratio between their values (Se-ANS/CRF-PNS%) was close to unity. There was no divergence of the middle part of the facial skeleton; the cranial and spinal planes were almost parallel. The antero-posterior size of the maxilla (A'-M', A'-PNS) was considerably smaller compared to the control group. The low value of the SSeA angle and posterior positioning of the maxilla were more severe cephalometric characteristics.

The prevalence of condylar growth and vertically insufficient height of the alveolar processes resulted in the anterior upward movement of the mandible. The ASeB angle significantly decreased in the period of mixed dentition, and had a negative value in the

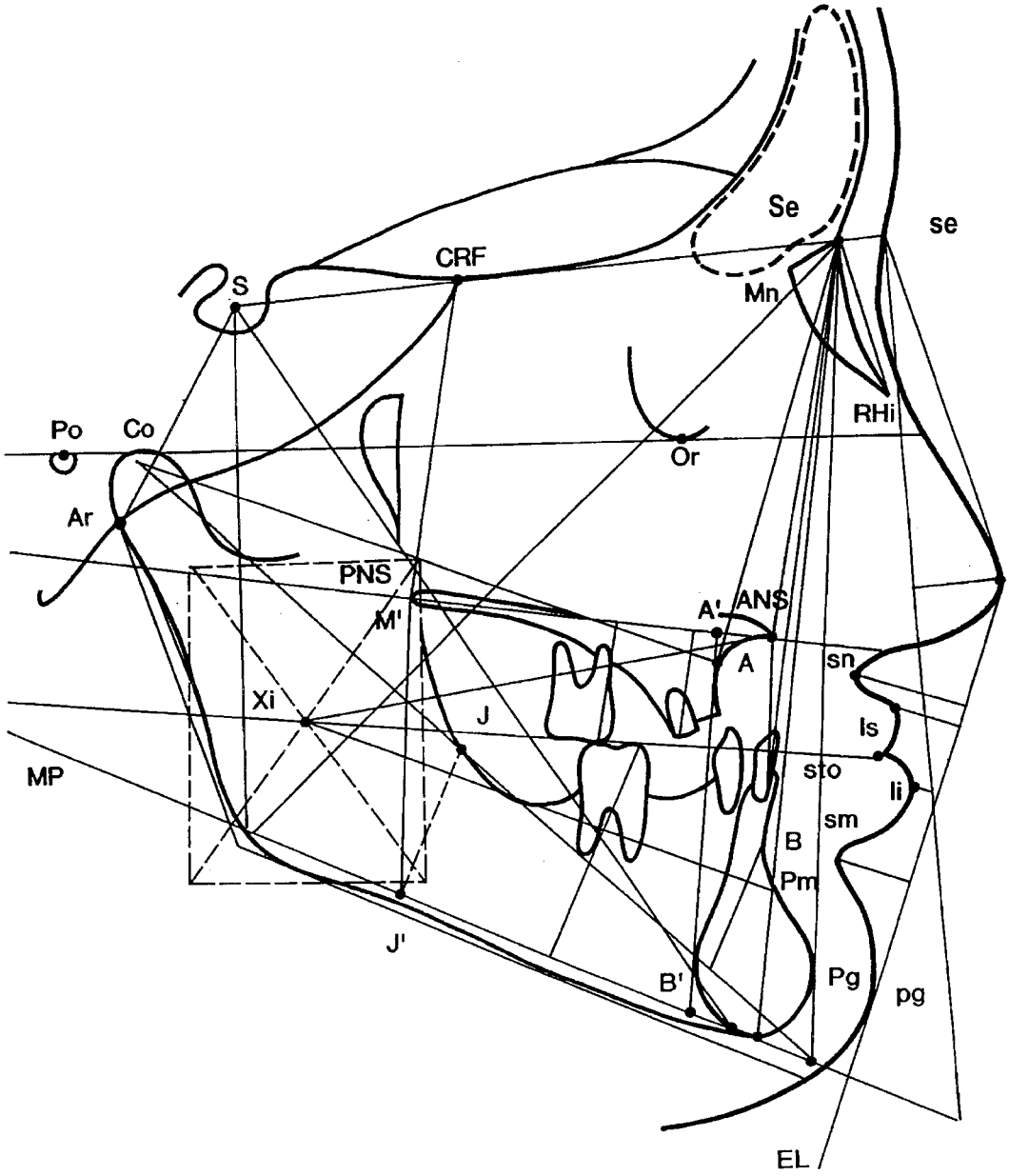

Fig. 1. Diagrammatic representation of the points digitized on each lateral skull radiograph. Defined points are listed in Table 1.

permanent dentition with severe hypodontia. These patients often demonstrated a mandibular prognathism in relation to the maxillary deficit and a tendency towards a class III malocclusion with anterior crossbite. The values of the mandibular plane inclination (<Se S Gn Go), Y-axis plane (< Se S Gn), lower gonial angle (<Se Go Gn), and Björk's sum angle (<Se S Ar + <S Ar Go + <Ar Go Gn), <Sp P MP, were much smaller compared to the control group. The lower anterior facial height (< ANS-Xi-Pm,

TABLE 1. Names and definitions of landmarks and planes in the maxilla and mandible

a. Point name	Definition
A	Maxillary A point: the deepest part of the concavity of the maxilla beneath the anterior nasal spine.
A′	A point on the maxillary plane projected perpendicular to the maxillary plane from the A point.
ANS	Anterior nasal spine.
Ar	Articulare: a point where the posterior outline of the condyle passes over the posterior and lower margin of the cranial base.
B	Mandibular B point: deepest part of the mandibular alveolar concavity.
B′	A point of projection of a perpendicular from the mandibular plane of the B point.
Co	Condylion: most posterior and superior part of the condylar head.
CRF	Cribriform: intersection of the greater wings of the sphenoid bone and the anterior cranial base.
Go	Gonion: the constructed most posterior and inferior point at the angle of the mandible.
Gn	Gnathion: the most inferior point of the mandible midway between Pg and Me.
Li	Most prominent point of the vermilion border of the lower lip.
Ls	Most prominent point of the vermilion border of the upper lip.
Me	Menton: the junction of the symphysis with the mandibular border.
Or	Orbitale: the most inferior anterior point on the border of the orbit.
Pg	Pogonion: the most anterior part of the bony chin.
pg	Soft-tissue pogonion: the most anterior point on the tip of the chin.
Pm	Pm point (Ricketts): anterior border of symphysis, halfway between B and Pg.
Pn	Pronasale: most prominent soft-tissue point on the tip of the nose.
PNS	Posterior nasal spine.
Po	Porion: the superior border of the bony external auditory meatus.
Rhi	Rhinion: tip of the nasal bone.
S	Sella: the central point of the sella turcica.
Se	Sellion: the innermost point on the concavity between the frontal and nasal bones. In younger patients, where the fronto-nasal suture is still patent, it is the most anterior point on the suture.
se	Soft-tissue sellion, reflecting the maximum concavity of the nasal bones, representing the soft-tissue equivalent of point Se.
Sn	Soft-tissue subnasale.
Sto	Stomion: the most anterior contact of upper and lower lips.

TABLE 1. (continued)

b. Line and plane name	Definition
EL	Esthetic line joining the tip of the soft-tissue profile of the nose to the soft-tissue outline of the pogonion (pg).
J	A line called the facial axis, joining Co to Pn.
J′	Perpendicular line reflecting midpoint of J line onto mandibular plane.
M′	Posterior facial height: from PNS perpendicular to maxillary plane to MP.
Mn	Axis joining se to G.
MP	Mandibular plane: a line joining Go with Me.

TABLE 2. Missing teeth

Tooth	Percent of missing teeth[1]
Maxillary lateral incisors	13.54
Mandibular second premolars	11.15
Maxillary second premolars	10.64
Mandibular central incisors	9.15

[1] Of an analysis of 1,516 consecutive patients referred for assessment, 16.9% (256 patients) had at least one congenitally absent missing permanent tooth. Maxillary teeth accounted for 51.35% of all absent teeth. There were no missing molars in the permanent dentition in group 4.

ANS-Me) was also considerably reduced due to deficiency in vertical dimension of the jaws. Defective alveolar bone growth was revealed in both jaws.

The posterior facial height (Ar-Go) was shorter only in children with hypodontia (groups 2 and 3). In adolescents, this dimension increased up to the average value. The ratio Ar-Go/ANS-Me% increased, resulting in hypo-divergence of the lower part of the face. The hard-tissue changes in facial morphology led to the typical "aged-face" appearance of the patients with severe hypodontia. A saddle-nose deformity with depressed root and flat bridge is due to retroposition on the nasal bones in HED patients (group 3). The dimension from the tip of the soft-tissue nose to the nasal plane (pn-Pn) and (Sn pn) angle were less than in the control group.

The facial profile was flat or concave ($<$se pn pg was increased up to 8.4 $\pm$ 1.56 in children and 16.81 $\pm$ 2.18 in adolescents). The retroposition of the upper jaw caused the distal position of the subnasion point (Sn) in relation to EL. The naso-labial angle

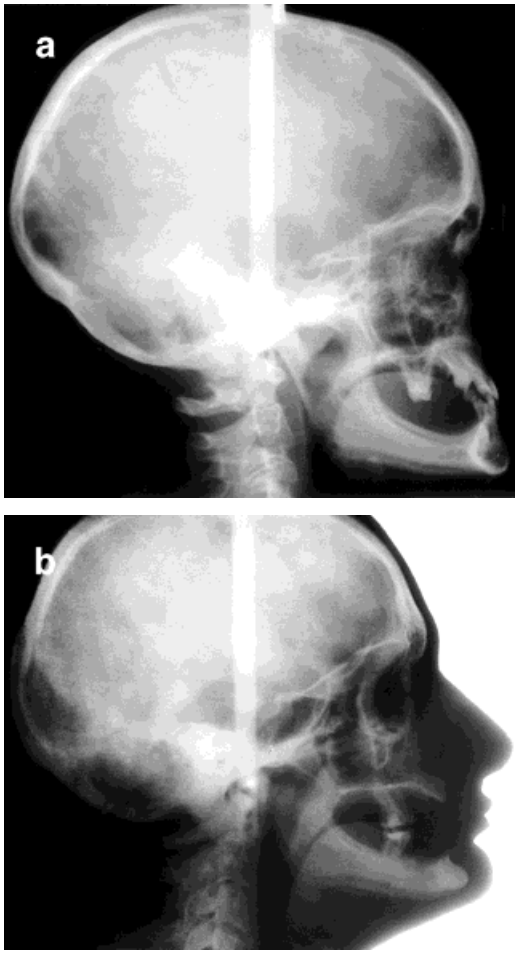

Fig. 2. Typical radiographs of patients suffering from HED. **a:** Clinically diagnosed hypodontia with the absence of 28 permanent teeth (patient age, 21 years), but without any systemic defects. **b:** The second patient (age 23) has 29 missing teeth but has the clinical signs of syndromic hypohidrotic ectodermal dysplasia.

was obtuse in group 2 patients when compared to nonaffected patients. In adolescents with HED, the naso-labial angle became acute, and the lips were protuberant and everted due to a markedly reduced vertical height of the lower face. The sulcus supramental was deeper than in nonaffected patients. The lips were behind the EL.

DISCUSSION

Although there is ample evidence that in anatomically modern humans, reduction in the facial skeleton is thought to be a consequence of changes in the consistency of the human diet (Kaifu, 1997), genetic phenomena unrelated to such an environmental influence also have a role in the reduction in facial height. Our study design allows the inference that as-yet unknown genetic mechanisms may be present to bring about the changes in the facial skeleton, just as known genetic conditions also have specific impacts on the facial skeleton.

As detailed earlier, it is now believed that absence of teeth can have a significant genetic basis. Two major types of HED are found: the hypohidrotic and hidrotic types (Bergsma, 1973). In the former there is sparseness of hair, absent or decreased sweat glands, and hypodontia; in the latter there are abnormalities of hair and nails, but teeth and sweat glands are normal. These two types of syndrome differ in their mode of inheritance: the hidrotic type is transmitted as an autosomal dominant trait, whereas the HED syndrome is transmitted as an X-linked recessive trait. Carrier females of syndromic HED can show mild signs of the disease such as sparse hair and patchy anhidrosis, together with an increased incidence of absence of teeth.

The prevalence of the absence of teeth has been shown to be 5.7% in females and 3.1% in males aged between 11–14 years (Brook, 1974). Further studies showed that when hypodontia of teeth existed, 80% of affected subjects had one or two missing teeth, while only 5% had five or more missing teeth (Silverman and Ackerman, 1979). In affected syndromic HED patients, the average number of missing teeth is reported to be 23.7 (Crawford et al., 1991). The pattern of hypodontia was similar to previous studies in that the first molars and upper central incisors were the teeth that were least affected by agenesis (Nakata et al., 1980).

In this study, we have demonstrated that both syndromic and nonsyndromic hypodontia cases are dependent on the teeth for growth in vertical height. Both the upper and lower facial heights were involved, and the differences were most noticeable in the mixed dentition when compared to unaffected patients. Here the overall facial height differed by 20.91 $\pm$ 1.94 mm in the mixed dentition of syndromic patients. It also demonstrates that

TABLE 3. A comparison of nonsyndromic patients with control patients (group 2 and group 1)[1]

	Mixed dentition		Permanent dentition	
Cephalometric values	D ± SD	*P*	D ± SD	*P*
Se-Me	−8.97 ± 1.58	<0.001		
S-Go	−4.42 ± 1.06	<0.001		
S-Go/Se-Me% ratio			4.61 ± 0.81	<0.01
ANS-Me	5.10 ± 1.23	<0.001	3.44 ± 1.01	<0.01
Ar-Go	−3.23 ± 0.87	<0.001		
Ar-Go/ANS-Me% ratio	5.13 ± 2.04	<0.05	9.53 ± 1.68	<0.001
<ANS Xi Pm	−4.13 ± 0.77	<0.001	−6.60 ± 0.77	<0.001
<SeS GoGn			−5.96 ± 1.16	<0.001
<Y-axis/<SeSGn/			−4.65 ± 0.99	<0.001

[1] D ± SD, mean difference ± standard deviation of the differences. *P*, probability. −, a reduction in the dimension/angle; lack of minus sign indicates an increase. For definitions of acronyms, see Table 1.

TABLE 4. A comparison of syndromic patients with control patients (group 3 and group 1)[1]

	Mixed dentition		Permanent dentition	
Cephalometric values	D ± SD	*P*	D ± SD	*P*
Se-Me	−20.91 ± 1.94	<0.001	−7.94 ± 1.41	<0.001
S-Go	−9.28 ± 1.40	<0.001		
S-Go/Se-Me% ratio	5.61 ± 1.25	<0.001	6.82 ± 1.14	<0.001
Se-ANS	−8.53 ± 0.92	<0.001		
CRF-PNS	−4.51 ± 0.92	<0.001		
CRF-PNS/Se-ANS% ratio	8.23 ± 1.67	<0.001	7.08 ± 1.61	<0.001
ANS-Me	12.89 ± 1.44	<0.001	−5.91 ± 1.32	<0.001
Ar-Go	−5.25 ± 1.11	<0.001		
Ar-Go/ANS-Me% ratio	11.62 ± 2.28	<0.001	11.40 ± 2.43	<0.001
<ANS Xi Pm	−8.66 ± 1.15	<0.001	−6.81 ± 1.10	<0.001
<SeS GnGo	−7.86 ± 1.48	<0.001	−10.10 ± 1.34	<0.001
<Y-axis/SeSGn/	−7.75 ± 1.04	<0.001	−8.42 ± 1.11	<0.001
<SseA	−7.47 ± 1.09	<0.001	−7.37 ± 1.17	<0.001
<SSeRhi	−4.97 ± 2.01	<0.05	−10.42 ± 1.75	<0.001

[1] D ± SD, mean difference ± standard deviation of the differences. *P*, probability. −, a reduction in the dimension/angle; lack of minus sign indicates an increase. For definitions of acronyms, see Table 1.

TABLE 5. A comparison of nonsyndromic patients with syndromic patients (group 2 and group 3)[1]

	Mixed dentition		Permanent dentition	
Cephalometric values	D ± SD	*P*	D ± SD	*P*
Se-Me	−11.94 ± 1.84	<0.001	−4.74 ± 1.41	<0.01
<SeS GoGn	−5.41 ± 1.15	<0.01	−4.15 ± 1.20	<0.01
Se-ANS	−5.69 ± 0.88	<0.001	−3.01 ± 0.94	<0.01
CRF.PMS/Se-ANS% ratio	5.99 ± 1.72	<0.01	5.80 ± 1.60	<0.01
<SeSSpP	−4.41 ± 0.82	<0.001	−6.41 ± 0.85	<0.001
<SseA			−3.38 ± 1.17	<0.01
Co-A	−4.46 ± 1.18	<0.01		
A-PNS	−2.84 ± 0.75	<0.01	−3.29 ± 0.82	<0.01
<Y-axis/SeSGn/	−6.58 ± 0.87	<0.001	−3.77 ± 1.02	<0.01
<Summ	−6.02 ± 1.34	<0.001	−4.37 ± 1.43	<0.01
<se pn pg	−6.20 ± 1.57	<0.01	−16.75 ± 4.85	<0.01
<SseRh	−5.43 ± 1.96	<0.01		

[1] D ± SD, mean difference ± standard deviation of the differences. *P*, probability. −, indicates a reduction in the dimension/angle; lack of minus sign indicates an increase. For definitions of acronyms, see Table 1.

growth in craniofacial height appears to be dependent on the alveolar bone processes.

This finding is also in agreement with previous work, which identified that facial height and maxillary length were reduced. The patient's profile was also found to be flattened, with lips well behind the expected profile, although the E line was not used in that study, in favor of the S-N-A point (Bixler et al., 1988).

However, in the case of syndromic HED patients, there appears to be a significant

reduction in forward growth of the nasal bones, with a typical "saddle-nosed deformity" and posterior positioning of the nasal bones. This seems to imply that some aspect of normal nasal growth and development appears deficient and that the control mechanisms of growth may be affected as a consequence of the underlying genetic basis of this disease. A candidate for causing this defect is the forward growth of the nose as a consequence of deficient cartilaginous growth of the nasal septum (Wexler and Sarnat, 1965). Furthermore, a possible hypothesis of partial penetration of the genetic disorder could be considered for the nonsyndromic HED patients. Analysis of this problem requires detailed pedigree analysis and examination of the nuclear material of affected individuals.

CONCLUSIONS

Patients with severe hypodontia present many oral problems. Specific aspects are common to non-HED edentulous patients, and some are unique to the HED condition. The two groups often have typical craniofacial characteristics and decreased occlusal vertical dimension as a consequence of limited alveolar bone. HED patients have a unique clinical problem expressed as abnormal craniofacial development and reduced mid-facial growth. This study presents the baseline data for the treatment of children with HED, showing their variations in growth and development. In addition, the evolutionary trend to decrease the size of the facial skeleton is seen to be further amplified by the absence of teeth.

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